WE CLAIM:

1. A method to enhance the cytotoxic activity of an antineoplastic drug against a disorder of abnormal cell proliferation, comprising administering an effective amount of the antineoplastic drug to a host in need of treatment in combination with an effective cytotoxicity-increasing amount of an antioxidant.

2. A method to decrease the toxicity to an antineoplastic agent administered for the treatment of a solid growth of abnormally proliferating cells, comprising administering an antioxidant prior to, with, or following the antineoplastic treatment.

3. A method to increase the therapeutic index of an antineoplastic agent administered for the treatment of a solid growth of abnormally proliferating cells, comprising administering an antioxidant prior to, with, or following the antineoplastic treatment.

4. A method to increase the nuclear localization of C/EBPβ in a cell, comprising administering an antioxidant to the interior of the cell.

5. A method to inhibit the carboxymethylation of the catalytic subunit of protein phosphatase 2A by the methyltransferase which acts on protein phosphatase 2A, comprising contacting methyltransferase with an antioxidant in a sufficient amount to achieve inhibition.

6. A method for the identification of compounds that increase the cytotoxicity of antineoplastic agents comprising assessing the compound's ability to promote phosphorylation at Ser^{299} of C/EBPβ.

7. A method for the identification of compounds that increase the cytotoxicity of antineoplastic agents comprising assessing the compound's ability to inhibit the carboxymethylation of protein phosphatase 2A.

8. A peptide sequence of the form -X1-Arg-X2-Ser-X3 (Sequence ID No. 2) wherein X2 is the C/EBP-β amino acid at position 298, and X1 and X3 represent flanking peptide sequences with substantial homology to C/EBPβ.

9. The method of claim 1, wherein the abnormal cell proliferation is colorectal cancer.

10. The method of claim 1, wherein the abnormal cell proliferation is breast cancer.

11. A protein complex that consists of C/EBPβ, PP2A, and the methyltransferase responsible for PP2A subunit carboxymethylation, in at least 70% purity.

12. A method for treating a condition of abnormal cell proliferation in a host, comprising administering to the host an effective amount of C/EBPβ, or a protein with substantial homology to C/EBPβ, in phosphorylated or unphosphorylated form.

13. The method of claim 13, wherein the protein with substantial homology to C/EBPβ consists of or contains a peptide sequence of the form -X1-Arg-X2-Ser-X3 (Sequence ID No. 2) wherein X2 is the C/EBPβ amino acid at position 298, and X1 and X3 represent flanking peptide sequences with substantial homology to those of C/EBPβ, and wherein the term substantial homology refers to a protein or peptide sequence that performs substantially the same function as the parent sequence and has at least 60% sequence identity.

14. A synthetic C/EBPβ analog that has a stabilized phosphate bond or an analog thereof that is resistant to dephosphorylation.

15. The synthetic analog of claim 15 that is a phosphoroamidate or phosphonate analog.

16. 16.The method of claim 1, 2, 3, 4 or 5, wherein the antioxidant is a dithiocarbamate.

17. The method of claim 16, wherein the dithiocarbamate is of the structure A-SC(S)-B, wherein A is hydrogen or a pharmaceutically acceptable cation, and B is alkyl, alkenyl, alkynyl, alkaryl, aralkyl, haloalkyl, haloalkenyl, haloalkynyl, aryl, alkaryl, hydrogen, C_{1-6} alkoxy-C_{1-10} alkyl, C_{1-6} alkylthio-C_{1-10} alkyl, NR^2R^3, $-(CHOH)_nCH_2OH$ (wherein n is 0, 1, 2, 3, 4, 5 or 6), $-(CH_2)_nCO_2R^1$, including alkylacetyl, alkylpropionyl and alkylbutyryl, or hydroxy(C_{1-6})alkyl- (wherein one or more hydroxyl groups are located on any of the carbon atoms), or NR^2R^3, wherein R^2 and R^3 are independently alkyl; $-(CHOH)_n(CH_2)_nOH$, wherein n is 0, 1, 2, 3, 4, 5 or 6; $-(CH_2)_nCO_2R^1$, $-(CH_2)_nCO_2R^4$; hydroxy(C_{1-6})alkyl-; alkenyl (including but not limited to vinyl, allyl, and $CH_3CH=CH-CH_2.CH_2$); alkyl(CO_2H), alkenyl(CO_2H), alkynyl(CO_2H), or aryl, wherein

the aryl group can be substituted as described above, notably, for example, with a NO_2, CH_3, t-butyl, CO_2H, halo, or p-OH group; or R^2 and R^3 can together constitute a bridge such as $-(CH_2)_m-$, wherein m is 3, 4, 5, 6, 7, 8, 9 or 10, and wherein R^4 is alkyl, aryl, alkaryl, or aralkyl, including acetyl, propionyl, and butyryl, or wherein B can be a heterocyclic or alkylheterocyclic group, which can be partially or totally hydrogenated.

18. The method of claim 1, 2, 3, 4, 5 or 6, wherein the antioxidant is probucol or a mono or diester thereof.

19. The method of claim 18, wherein one or both of the hydroxyl groups of probucol are replaced with esters of succinic acid, glutaric acid, adipic acid, suberic acid, sebacic acid, azelaic acid or maleic acid.

20. The method of claim 1, 2, 3, 4, 5 or 6 wherein the antioxidant is a 2,6-dialkyl-4-silylphenol.

21. The method of claim 1, 2, 3, 4, 5 or 6, wherein the antioxidant is N-acetyl cysteine.

22. The method of claim 1, 2, 3, 4, 5 or 6, wherein the antioxidant is selected from the group consisting of a scavenger of peroxide, a thiol, an inhibitor of lipid peroxidation, a dietary antioxidant, inhibitors of lipoxygenases and cyclooxygenases, antioxidants manufactured by the body, and synthetic phenolic antioxidants.

23. The method of claim 1, 2, 3, 6 or 7, wherein the antineoplastic agent is selected from the group consisting of Aceglatone; Aclarubicin; Altretamine; Aminoglutethimide; 5-Aminogleavulinic Acid; Amsacrine; Anastrozole; Ancitabine Hydrochloride; 17-1A Antibody; Antilymphocyte Immunoglobulins; Antineoplaston A10; Asparaginase; Pegaspargase; Azacitidine; Azathioprine; Batimastat; Benzoporphyrin Derivative; Bicalutamide; Bisantrene Hydrochloride; Bleomycin Sulphate; Brequinar Sodium; Broxuridine; Busulphan; Campath-IH; Caracemide; Carbetimer; Carboplatin; Carboquone; Carmofur; Carmustine; Chlorambucil; Chlorozotocin; Chromomycin; Cisplatin; Cladribine; Corynebacterium parvum; Cyclophosphamide; Cyclosporin; Cytarabine; Dacarbazine; Dactinomycin; Daunorubicin Hydrochloride; Decitabine; Diaziquone; Dichlorodiethylsulphide; Didemnin B.; Docetaxel; Doxifluridine; Doxorubicin Hychloride; Droloxifene; Echinomycin; Edatrexate; Elliptinium;

Elmustine; Enloplatin; Enocitabine; Epirubicin Hydrochloride; Estramustine Sodium Phosphate; Etanidazole; Ethoglucid; Etoposide; Fadrozole Hydrochloride; Fazarabine; Fenretinide; Floxuridine; Fludarabine Phosphate; Fluorouracil; Flutamide; Formestane; Fotemustine; Gallium Nitrate; Gencitabine; Gusperimus; Homoharringtonine; Hydroxyurea; Idarubicin Hydrochloride; Ifosfamide; Ilmofosine; Improsulfan Tosylate; Inolimomab; Interleukin-2; Irinotecan; JM-216; Letrozole; Lithium Gamolenate; Lobaplatin; Lomustine; Lonidamine; Mafosfamide; Melphalan; Menogaril; Mercaptopurine; Methotrexate; Methotrexate Sodium; Miboplatin; Miltefosine; Misonidazole; Mitobronitol; Mitoguazone Dihydrochloride; Mitolactol; Mitomycin; Mitotane; Mitozanetrone Hydrochloride; Mizoribine; Mopidamol; Multialchilpeptide; Muromonab-CD3; Mustine Hydrochloride; Mycophenolic Acid; Mycophenolate Mofetil; Nedaplatin; Nilutamide; Nimustine Hydrochloride; Oxaliplatin; Paclitaxel; PCNU; Penostatin; Peplomycin Sulphate; Pipobroman; Pirarubicin; Piritrexim Isethionate; Piroxantrone Hydrochloride; Plicamycin; porfimer Sodium; Prednimustine; Procarbazine Hydrochloride; Raltitrexed; Ranimustine; Razoxane; Rogletimide; Roquinimex; Sebriplatin; Semustine; Sirolimus; Sizofiran; Sobuzoxane; Sodium Bromebrate; Sparfosic Acid; Sparfosate Sodium; Sreptozocin; Sulofenur; Tacrolimus; Tamoxifen; Tegafur; Teloxantrone Hydrochloride; Temozolomide; Teniposide; Testolactone; Tetrasodium Mesotetraphenylporphine-sulphonate; Thioguanine; Thioinosine; Thiotepa; Topotecan; Toremifene; Treosulfan; Trimetrexate; Trofosfamide; Tumor Necrosis Factor; Ubenimex; Uramustine; Vinblastine Sulphate; Vincristine Sulphate; Vindesine Sulphate; Vinorelbine Tartrate; Vorozole; Zinostatin; Zolimomab Aritox; and Zorubicin Hydrochloride.

24. The method of claim 1, 2, 3 or 12, wherein the abnormal cell proliferation is a benign tumor.

25. The method of claim 1, 2, 3 or 12, wherein the abnormal cell proliferation is a malignant tumor.

26. The method of claim 1, 2, 3 or 12, wherein the abnormal cell proliferation is a hyperproliferative or preneoplastic lesion.

27. The method of claim 1, 2, 3 or 12, wherein the abnormal cell proliferation is selected from the group consisting of papilloma, adenoma, firoma, chondroma, osteoma, lipoma, hemangioma, lymphangioma, leiomyoma, rhabdomyoma, meningioma, neuroma, ganglioneuroma, nevus, pheochromocytoma, neurilemona, fibroadenoma, teratoma, hydatidiform mole, granuosa-theca, Brenner tumor, arrhenoblastoma, hilar cell tumor, sex cord mesenchyme, interstitial cell tumor, thyoma, renal cell carcinoma, prostatic adenocarcinoma, bladder carcinoma,adenocarcinoma, fibrosarcoma, chondrosarcoma, osteosarcoma, liposarcoma, hemangiosarcoma, lymphangiosarcoma, leiomyosarcoma, rhabdomyosarcoma, myelocytic leukemia, erythroleukemia, multiple myeloma, glioma, meningeal sarcoma, thyoma, cystosarcoma phyllodes, nephroblastoma, teratoma choriocarcinoma, cutaneous T-cell lymphoma (CTCL), cutaneous tumors primary to the skin or infiltrating the skin, Kaposi's sarcoma, and premalignant and malignant diseases of mucosal tissues, central nervous system tumors, mycosis fungoides, psoriasis, dermatomyositis, rheumatoid arthritis, viruses, molluscum contagiosum, remalignant and malignant diseases of the female genital tract.

28. The method of claim 1, 2, 3 or 12, wherein the abnormal cell proliferation is selected from the group consisting of colorectal cancer, ovarian cancer, bone cancer, renal cancer, breast cancer, gastric cancer, pancreatic cancer, melanoma and hematopoietic tumors.

29. The method of claim 1, 2, 3 or 12, wherein the abnormal cell proliferation is a cardiovascular condition.

30. The method of claim 29, wherein the cardiovascular condition is post angioplasty restenosis.